WEST Search History

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			- Odilooi

DATE: Thursday, March 09, 2006

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	DB=PC	GPB,USPT,DWPI; PLUR=YES; OF	P=ADJ
£"`	L2	11 and (cd63 near5 macrophag\$)	9
8011	Τ1	cd63 and antibod®	651

END OF SEARCH HISTORY

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FILE 'HOME' ENTERED AT 14:48:47 ON 09 MAR 2006

=> f medline biosis CA embase scisearch
THIS COMMAND NOT AVAILABLE IN THE CURRENT FILE
Some commands only work in certain files. For example, the EXPAND
command can only be used to look at the index in a file which has an
index. Enter "HELP COMMANDS" at an arrow prompt (=>) for a list of
commands which can be used in this file.

=> f medline; f biosis; f CA; f embase; f scisearch THIS COMMAND NOT AVAILABLE IN THE CURRENT FILE COMMAND STACK INTERRUPTED. ENTER "DISPLAY HISTORY" TO SEE WHICH COMMANDS WERE EXECUTED.

Some commands only work in certain files. For example, the EXPAND command can only be used to look at the index in a file which has an index. Enter "HELP COMMANDS" at an arrow prompt (=>) for a list of commands which can be used in this file.

=> f medline

THIS COMMAND NOT AVAILABLE IN THE CURRENT FILE Some commands only work in certain files. For example, the EXPAND command can only be used to look at the index in a file which has an index. Enter "HELP COMMANDS" at an arrow prompt (=>) for a list of commands which can be used in this file.

=> FIL BIOSIS

COST IN U.S. DOLLARS .

SINCE FILE TOTAL ENTRY SESSION 0.84 0.84

1.71

FULL ESTIMATED COST

FILE 'BIOSIS' ENTERED AT 14:51:20 ON 09 MAR 2006 Copyright (c) 2006 The Thomson Corporation

FILE COVERS 1969 TO DATE.

CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 9 March 2006 (20060309/ED)

=> f embase

L1 1327 EMBASE

=> fil medline; fil biosis; fil CA; fil Embase; fil Scisearch
COST IN U.S. DOLLARS
SINCE FILE TOTAL
ENTRY SESSION

FULL ESTIMATED COST 0.87

FILE 'MEDLINE' ENTERED AT 14:52:04 ON 09 MAR 2006

FILE LAST UPDATED: 8 MAR 2006 (20060308/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>). See also:

http://www.nlm.nih.gov/mesh/

http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html

http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html

http://www.nlm.nih.gov/pubs/techbull/nd05/nd05 2006 MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the

MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

COST IN U.S. DOLLARS

SINCE FILE

ENTRY SESSION

TOTAL

FULL ESTIMATED COST

0.39 2.10

FILE 'BIOSIS' ENTERED AT 14:52:04 ON 09 MAR 2006 Copyright (c) 2006 The Thomson Corporation

FILE COVERS 1969 TO DATE.

CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 9 March 2006 (20060309/ED)

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

FULL ESTIMATED COST

ENTRY

0.87

SESSION 2.97

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FILE COVERS 1907 - 2 Mar 2006 VOL 144 ISS 11 FILE LAST UPDATED: 2 Mar 2006 (20060302/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

FULL ESTIMATED COST

ENTRY SESSION

0.44 3.41

FILE 'EMBASE' ENTERED AT 14:52:04 ON 09 MAR 2006 Copyright (c) 2006 Elsevier B.V. All rights reserved.

FILE COVERS 1974 TO 3 Mar 2006 (20060303/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

COST IN U.S. DOLLARS

SINCE FILE

ENTRY SESSION

FULL ESTIMATED COST

1.43

4.84

TOTAL

'FILE 'SCISEARCH' ENTERED AT 14:52:04 ON 09 MAR 2006 Copyright (c) 2006 The Thomson Corporation

FILE COVERS 1974 TO 2 Mar 2006 (20060302/ED)

SCISEARCH has been reloaded, see HELP RLOAD for details.

=> cd63

CD63 IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system. For a list of commands available to you in the current file, enter "HELP COMMANDS" at an arrow prompt (=>).

=> s cd63

L2 716 CD63

=> d l2 ibib abs 1-5

L2 ANSWER 1 OF 716 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on

STN

ACCESSION NUMBER: 2006:181023 SCISEARCH

THE GENUINE ARTICLE: 012HV

TITLE: Trafficking from CD63-positive late endocytic

multivesicular bodies is essential for intracellular

development of Chlamydia trachomatis

AUTHOR: Beatty W L (Reprint)

CORPORATE SOURCE: Washington Univ, Sch Med, Dept Mol Microbiol, St Louis, MO

63110 USA (Reprint) beatty@borcim.wustl.edu

COUNTRY OF AUTHOR: U

SOURCE: JOURNAL OF CELL SCIENCE, (15 JAN 2006) Vol. 119, No. 2,

pp. 350-359. ISSN: 0021-9533.

PUBLISHER: COMPANY OF BIOLOGISTS LTD, BIDDER BUILDING CAMBRIDGE

COMMERCIAL PARK COWLEY RD, CAMBRIDGE CB4 4DL, CAMBS,

ENGLAND.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English REFERENCE COUNT: 47

ENTRY DATE: Entered STN: 23 Feb 2006

Last Updated on STN: 23 Feb 2006

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Chlamydiae are obligate intracellular bacterial pathogens that replicate solely within the confines of a membrane-bound vacuole termed an inclusion. Within this protected organelle, chlamydiae acquire host-cell-derived biosynthetic precursors necessary for intracellular subsistence, yet the mechanisms and pathways responsible for this acquisition remain elusive. The present study identifies an interaction between the chlamydial inclusion and multivesicular bodies, complex organelles pivotal in protein and lipid transport that are positioned along the endosome-lysosome pathway, and intersect the exocytic pathway in various cell types. Resident protein and lipid constituents of multivesicular bodies colocalized with intracellular chlamydiae, with direct delivery of the resident protein CD63 to the chlamydial inclusion. Interruption of trafficking from multivesicular bodies by pharmacological inhibitors and exogenous antibodies subsequently disrupted sphingolipid delivery to the maturing chlamydial inclusion and intracellular bacterial growth. This study identifies a trafficking pathway from CD63-positive multivesicular bodies to the bacterial inclusion, a novel interaction that provides essential lipids necessary for maintenance of a productive intracellular infection.

L2 ANSWER 2 OF 716 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2006:162126 SCISEARCH

THE GENUINE ARTICLE: 009LK

TITLE: Activation of blood platelets in echinococcosis - CD62P

and CD63 expression

· AUTHOR: Matowicka-Karna J (Reprint); Kemona H; Dymicka-Piekarska

V; Butkiewicz A

CORPORATE SOURCE: Med Univ Bialystok, Dept Clin Lab Diagnost, J Waszyngtona

> 15A, PL-15274 Bialystok, Poland (Reprint); Med Univ Bialystok, Dept Clin Lab Diagnost, PL-15274 Bialystok,

Poland

matowic@amb.edu.pl

COUNTRY OF AUTHOR:

Poland

PARASITOLOGY RESEARCH, (FEB 2006) Vol. 98, No. 3, pp. SOURCE:

214-217.

ISSN: 0932-0113.

SPRINGER, 233 SPRING STREET, NEW YORK, NY 10013 USA. PUBLISHER:

Article; Journal DOCUMENT TYPE:

LANGUAGE:

English

REFERENCE COUNT:

30

ENTRY DATE:

Entered STN: 16 Feb 2006

Last Updated on STN: 16 Feb 2006

L2ANSWER 3 OF 716 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on

STN

ACCESSION NUMBER: 2006:122429 SCISEARCH

THE GENUINE ARTICLE: 007NO

TITLE:

Bacitracin reveals a role for multiple thiol isomerases in

platelet function

AUTHOR: Robinson A; O'Neill S; Kiernan A S; O'Donoghue N; Moran N

(Reprint)

CORPORATE SOURCE: Royal Coll Surgeons Ireland, Dept Clin Pharmacol, 123 St

Stephens Green, Dublin 2, Ireland (Reprint); Royal Coll Surgeons Ireland, Dept Clin Pharmacol, Dublin 2, Ireland

nmoran@rcsi.ie

COUNTRY OF AUTHOR:

Ireland

SOURCE: BRITISH JOURNAL OF HAEMATOLOGY, (FEB 2006) Vol. 132, No.

> 3, pp. 339-348. ISSN: 0007-1048.

PUBLISHER: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DO,

OXON, ENGLAND.

DOCUMENT TYPE: Article; Journal

LANGUAGE:

English 39

REFERENCE COUNT: ENTRY DATE:

Entered STN: 9 Feb 2006

Last Updated on STN: 9 Feb 2006

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The platelet-specific integrin alpha IIb beta 3 has endogenous thiol isomerase activity associated with the CXXC motifs within the beta subunit. Using a highly purified form of bacitracin, a thiol isomerase inhibitor, we now provide further evidence of the functional significance of this enzymatic activity in integrin activation. In addition, we demonstrate a role for multiple thiol isomerases in platelet function. This bacitracin prevented platelet aggregation to thrombin and collagen, and directly inhibited alpha IIb beta 3 activation, as detected by PAC-1 In parallel, bacitracin inhibited the endogenous thiol isomerase activity of purified alpha IIb beta 3 with a 50% inhibitory concentration of 15(.)5 mu mol/l. In order to determine whether the effects of bacitracin are solely mediated by inhibition of integrin enzymatic activity, we examined integrin-independent indices of platelet activation. We found bacitracin inhibited both platelet secretion (CD62P and CD63) and thromboxane (TxA(2)) production, with complete inhibition at different concentrations. Thus, we demonstrated a role for multiple thiol isomerases in platelet function. Taken together, these studies support a role for the endogenous integrin thiol isomerase activity in activation of alpha IIb beta 3 and highlight the novel regulation of platelet function by other, as yet undefined thiol isomerases.

L2ANSWER 4 OF 716 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2006:119755 SCISEARCH

THE GENUINE ARTICLE: 005VX

TITLE: Diagnosis of neuromuscular blocking agent hypersensitivity

reactions using cytofluorimetric analysis of basophils

AUTHOR: Kvedariene V; Kamey S; Ryckwaert Y; Rongier M; Bousquet J;

Demoly P; Arnoux B (Reprint)

CORPORATE SOURCE: Hop Arnaud Villeneuve, INSERM, U454, IFR3, F-34295

Montpellier 5, France (Reprint); Hop Arnaud Villeneuve, INSERM, U454, F-34295 Montpellier 5, France; Vilnius Univ

Hosp Santariskiu Klin, Vilnius, Lithuania; Hop A

Villeneuve, Unite Explorat Allergies, Montpellier, France; Kyoto Prefectural Univ Med, Kyoto, Japan; Hop Lapeyronie,

Dept Anesthesiol, Montpellier, France

COUNTRY OF AUTHOR: France; Lithuania; Japan

SOURCE: ALLERGY, (MAR 2006) Vol. 61, No. 3, pp. 311-315.

ISSN: 0105-4538.

PUBLISHER: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ,

OXON, ENGLAND.

DOCUMENT TYPE: Article; Journal

LANGUAGE:

English 28

REFERENCE COUNT: ENTRY DATE:

Entered STN: 9 Feb 2006

Last Updated on STN: 9 Feb 2006

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS AB Background: Immunoglobulin E (IgE) -mediated hypersensitivity reactions to neuromuscular blocking agents (NMBA) are common and life threatening. Basophil activation based upon the expression of CD63 in the presence of specific allergens was found to be of importance for the diagnosis of IgE-mediated hypersensibility.

Methods: The Basotest((R)) was evaluated for the diagnosis of NMBA in 47 patients with proven NMBA anaphylaxis, 40 atopic subjects nonallergic to NMBA and five healthy volunteers. Diagnosis of NMBA was made according to international standards on clinical history, skin tests and provocation tests when needed.

Results: In the NMBA allergic patients, sensitivity of Basotest((R)) was 36.1%, but it increased to 85.7% for reactions which occurred within the last 3 years. The specificity was 93.3%.

Conclusion: Basotest((R)) may be useful for the diagnosis of NMBA allergy in patients with a suspicion of recent IgE-mediated hypersensitivity reaction to NMBA.

ANSWER 5 OF 716 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on L2STN

ACCESSION NUMBER: 2006:119753 SCISEARCH

THE GENUINE ARTICLE: 005VX

TITLE:

Basophil allergen threshold sensitivity: a useful approach

to anti-IgE treatment efficacy evaluation

AUTHOR: Nopp A (Reprint); Johansson S G O; Ankerst J; Bylin G;

Cardell L O; Gronneberg R; Irander K; Palmqvist M; Oman H

CORPORATE SOURCE: Karolinska Univ Hosp L204, Dept Med, Allergy & Clin

Immunol Unit, S-17126 Stockholm, Sweden (Reprint); Karolinska Inst, Dept Med, Allergy & Clin Immunol Unit, Stockholm, Sweden; Karolinska Univ Hosp, Dept Clin Immunol & Transfus Med, Stockholm, Sweden; Univ Lund Hosp, Dept Med, S-22185 Lund, Sweden; Karolinska Univ Hosp Huddinge, Dept Med, Div Resp Med & Allergol, Stockholm, Sweden; Malmo Univ Hosp, Lab Clin Expt Allergy Res, Malmo, Sweden;

Karolinska Univ Hosp, Dept Resp Med, Allergy Sect, Stockholm, Sweden; Univ Hosp, Allergy Ctr, Linkoping, Sweden; Sahlgrenska Univ Hosp, Lung Pharmacol Grp,

Gothenburg, Sweden; MIAB, Uppsala, Sweden

COUNTRY OF AUTHOR:

Sweden SOURCE:

ALLERGY, (MAR 2006) Vol. 61, No. 3, pp. 298-302.

ISSN: 0105-4538.

PUBLISHER: BLACKWELL PUBLISHING, 9600 GARSINGTON RD, OXFORD OX4 2DQ,

OXON, ENGLAND.

DOCUMENT TYPE:

Article; Journal

LANGUAGE:

English

13

REFERENCE COUNT: ENTRY DATE:

Entered STN: 9 Feb 2006

Last Updated on STN: 9 Feb 2006

Background: Monitoring of the allergen sensitivity of a patient is most important for optimal patient care and a basic prerequisite for immunomodulating treatment. The objective of this study was to investigate how basophil allergen sensitivity can be applied in the monitoring of anti-immunoglobulin E (IgE) treatment.

Methods: Basophils from timothy grass pollen allergic patients were, by flow cytometry, analysed for allergen threshold sensitivity (CD-sens) by measuring CD63 up-regulation on CD203c-identified basophils. The results were compared with maximal percentage CD63 up-regulation at one allergen dose (CD-max), skin prick test end-point allergen titration, (SPT-sens), nasal provocation titration tests (nasal provocation titre) and serum IgE and IgE antibody concentrations.

Results: There was a significant correlation (r = 0.50, P = 0.01) between CD-sens and SPT-sens, CD-sens and the IgE antibody concentration in percentage of 'total IgE' (relative IgE antibody concentration) (r = 0.72, P < 0.001) as well as between CD-sens and nasal provocation titre (r = 0.54, P < 0.05) but, in contrast, CD-max did not correlate with any of the sensitization parameters, i.e. SPT-sens, nasal provocation titre, absolute and relative IgE antibody concentration or CD-sens. CD-sens could be used to monitor omalizumab treatment efficacy while, based on CD-max, four of seven symptom-free patients on omalizumab would have been classified as having ongoing allergy.

Conclusions: CD-sens seems to be very useful for the determination of a patient's allergen sensitivity and should be evaluated for the measurement and monitoring of anti-IgE treatment efficacy. CD-max, the conventional approach to basophil allergen challenge, which mirrors cell reactivity, gives incorrect information.

=> d his

L1

1.2

(FILE 'HOME' ENTERED AT 14:48:47 ON 09 MAR 2006)

FILE 'BIOSIS' ENTERED AT 14:51:20 ON 09 MAR 2006 1327 F EMBASE

FILE 'MEDLINE' ENTERED AT 14:52:04 ON 09 MAR 2006

FILE 'BIOSIS' ENTERED AT 14:52:04 ON 09 MAR 2006

FILE 'CA' ENTERED AT 14:52:04 ON 09 MAR 2006

FILE 'EMBASE' ENTERED AT 14:52:04 ON 09 MAR 2006

FILE 'SCISEARCH' ENTERED AT 14:52:04 ON 09 MAR 2006 716 S CD63

=> fil medline biosis CA Embase Scisearch COST IN U.S. DOLLARS

SINCE FILE TOTAL ENTRY SESSION 38.33 43.17

FULL ESTIMATED COST

FILE 'MEDLINE' ENTERED AT 14:53:54 ON 09 MAR 2006

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```
=> s cd63
- L3
           3599 CD63
/ => s hiv
 T.4
         575282 HIV
 => s hiv?
         700372 HIV?
 1.5
 => s 13 (p) 15
 L<sub>6</sub>
             93 L3 (P) L5
 => dup rem 16
 PROCESSING COMPLETED FOR L6
 L7
              28 DUP REM L6 (65 DUPLICATES REMOVED)
 => s 17 and py<=2000
    1 FILES SEARCHED...
               6 L7 AND PY<=2000
 1.8
 => d 18 ibib abs 1-6
 1.8
      ANSWER 1 OF 6
                        MEDLINE on STN
 ACCESSION NUMBER:
                      1998099250
                                     MEDLINE
 DOCUMENT NUMBER:
                      PubMed ID: 9438413
 TITLE:
                      Enhanced activation of platelets with abnormal release of
                      RANTES in human immunodeficiency virus type 1 infection.
 AUTHOR:
                      Holme P A; Muller F; Solum N O; Brosstad F; Froland S S;
                      Aukrust P
 CORPORATE SOURCE:
                      Research Institute for Internal Medicine, Medical
                      Department A, The National Hospital, University of Oslo,
 SOURCE:
                      The FASEB journal : official publication of the Federation
                      of American Societies for Experimental Biology, (1998
                      Jan) Vol. 12, No. 1, pp. 79-89.
                      Journal code: 8804484. ISSN: 0892-6638.
 PUB. COUNTRY:
                     United States
 DOCUMENT TYPE:
                     Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE:
                     English
 FILE SEGMENT:
                     Priority Journals; AIDS
 ENTRY MONTH:
                     199802
 ENTRY DATE:
                     Entered STN: 19980224
                     Last Updated on STN: 19980224
                     Entered Medline: 19980209
 AB
      Besides their role in hemostasis, platelets are involved in inflammatory
      and immunological processes, and we hypothesize that platelet activation
      may play an immunopathogenetic role in HIV-1 infection. Blood
      was drawn from 15 controls and 20 HIV-1-infected patients with
      normal platelet counts, classified into groups of non-AIDS and AIDS.
      Platelet activation was detected using flow cytometry with mAbs against
      the release markers P-selectin and CD63, mAb against GPIb, and
      the probe annexin V detecting surface exposure of aminophospholipids.
      amount of microvesicles was measured using mAb against GPIIIa.
      to controls, blood samples from HIV-1-infected patients showed
      significantly enhanced levels of microvesicles and activated platelets as
      detected by their exposure of P-selectin, CD63, and
      aminophospholipids, as well as reduction in GPIb expression. Increased
      expression of P-selectin and amounts of microvesicles were most pronounced
      in advanced clinical and immunological disease. When studying the effect
      of HIV-1 protease inhibitor therapy (indinavir) on platelet
      activation, we found that concomitant with a profound decrease in plasma
      viral load, there was a near normalization of several of the parameters
      reflecting enhanced platelet activation. Finally, we demonstrated that
      platelets may be an important source of the chemokine RANTES in
      HIV-1-infected patients. Although both unstimulated and
```

SFLLRN-stimulated platelets from asymptomatic patients had enhanced release of RANTES, platelets from AIDS patients were characterized by markedly enhanced spontaneous, but decreased SFLLRN-stimulated release of this chemokine. Taken together, these results, which demonstrate for the

first time increased platelet activation in HIV-1-infected patients with normal platelet counts, may represent a previously unrecognized immunopathogenic factor in HIV-1 infection.

ANSWER 2 OF 6 MEDLINE on STN ACCESSION NUMBER: 97271317 MEDLINE DOCUMENT NUMBER: PubMed ID: 9126268

TITLE: Cell membrane vesicles are a major contaminant of gradient-enriched human immunodeficiency virus type-1

preparations.

AUTHOR: Gluschankof P; Mondor I; Gelderblom H R; Sattentau Q J CORPORATE SOURCE:

Centre d'immunologie de Marseille-Luminy, France..

gluschan@ciml.univ-mrs.fr

SOURCE: Virology, (1997 Mar 31) Vol. 230, No. 1, pp.

125-33.

Journal code: 0110674. ISSN: 0042-6822.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals; AIDS

199706 ENTRY MONTH:

ENTRY DATE: Entered STN: 19970709

> Last Updated on STN: 19970709 Entered Medline: 19970626

AB During preliminary experiments to establish the proportion of virus-coded p24 protein to virus membrane-associated HLA-DR in gradient-enriched HIV-1 preparations, we became aware of a large variability between experiments. In order to determine whether HLA-DR-containing cellular material was contaminating the virus preparations, we carried out enrichment by gradient centrifugation of clarified supernatants from noninfected cells and tested this material for HLA-DR content. We found that, independently of the cell type used, gradient enrichment resulted in the isolation of large quantities of HLA-DR-containing material which banded at a density overlapping that of infectious HIV. Electron microscopy of gradient-enriched preparations from supernatants of virus-infected cells revealed an excess of vesicles with a size range of about 50-500 nm, as opposed to a minor population of virus particles of about 100 nm. Electron micrographs of infected cells showed polarized vesiculation of the cell membrane, and virus budding was frequently

colocalized with nonviral membrane vesiculation. Analysis of the cellular molecules present in the fractions containing virus or exclusively cellular material demonstrated that virus and cellular vesicles share several cellular antigens, with the exception of CD43 and CD63, found mainly at the virus surface, and HLA-DQ, which was found only in the cellular vesicles.

1.8 ANSWER 3 OF 6 MEDLINE on STN ACCESSION NUMBER: 94145751 MEDLINE DOCUMENT NUMBER: PubMed ID: 8312057

TITLE: Association of host cell surface adhesion receptors and

other membrane proteins with HIV and SIV.

AUTHOR: Orentas R J; Hildreth J E

CORPORATE SOURCE: Department of Pharmacology and Molecular Sciences, Johns

Hopkins University School of Medicine, Baltimore, Maryland

21205.

CONTRACT NUMBER: 5 R01 AI 31806 (NIAID) 5 T32 CA 09243 (NCI)

SOURCE: AIDS research and human retroviruses, (1993 Nov)

Vol. 9, No. 11, pp. 1157-65.

Journal code: 8709376. ISSN: 0889-2229.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals; AIDS

ENTRY MONTH: 199403

ENTRY DATE: Entered STN: 19940330

Last Updated on STN: 19970203 Entered Medline: 19940318

AB We have developed a MAb-based capture assay to study the association of

host cell membrane proteins with HIV and SIV. Class I and II MHC proteins were found to be associated with HIV as previously described. In addition to these molecules a number of other host molecules were found to be acquired by HIV, including CD71, CD63, CD43, and CD8. We have demonstrated that the major leukocyte adhesion receptors LFA-1 (CD11A/CD18) and CD44 are also associated with HIV. The level of surface expression of host membrane proteins did not predict relative expression (capture efficiency) of the virus. The use of virus-susceptible indicator cells showed that the assay involved host membrane protein-mediated capture of infectious HIV and SIV particles. Our data indicate that HIV and SIV acquire a number of host membrane proteins including adhesion receptors and that this process may be nonrandom. The acquisition of host cell adhesion receptors by HIV and SIV could have profound effects on the biology of the viruses, including binding, infectivity, and tropism.

L8 ANSWER 4 OF 6 MEDLINE on STN
ACCESSION NUMBER: 93139775 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8093711

TITLE: Host cell membrane proteins on human immunodeficiency virus

type 1 after in vitro infection of H9 cells and blood mononuclear cells. An immuno-electron microscopic study. Meerloo T; Sheikh M A; Bloem A C; de Ronde A; Schutten M; van Els C A; Roholl P J; Joling P; Goudsmit J; Schuurman H

J

CORPORATE SOURCE: Department of Pathology, University Hospital, Utrecht, The

Netherlands.

SOURCE: The Journal of general virology, (1993 Jan) Vol.

74 (Pt 1), pp. 129-35.

Journal code: 0077340. ISSN: 0022-1317.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals; AIDS

ENTRY MONTH: 199302

AUTHOR:

ENTRY DATE: Entered STN: 19930312

Last Updated on STN: 19970203 Entered Medline: 19930222

AB Human immunodeficiency virus type 1 (HIV-1)-infected H9 and blood mononuclear cells (MNCs) were studied by immunogold electron microscopy for the presence of HIV-1 gag p24 protein, env gp41 and gp120 proteins, and host cell molecules CD4, CD11a, CD25, CD54, CD63, HLA class I and HLA-DR. Uninfected H9 cells and MNC membranes labelled for CD4, HLA class I and class II, and, at low density, CD11a and CD54; lysosomal structures in the cytoplasm labelled for CD63. The infected cell surface showed immunolabelling for HIV-1 proteins, as did budding particle-like structures. Immunogold labelling of the cell membrane for CD4 was almost non-existent. The level of immunolabelling for CD11a and CD54 on infected cells was greater than that on uninfected cells; this is presumably related to a state of activation during virus synthesis. Budding particle-like structures and free virions in the intercellular space were immunogold-labelled for all host cell markers investigated. confirmed by double immunogold labelling using combinations of HIV -1 gag p24 labelling and labelling for the respective host cell molecule. We conclude that virions generated in HIV-1-infected cells concentrate host-derived molecules on their envelope. Also molecules with a prime function in cellular adhesion concentrate on the virion.

L8 ANSWER 5 OF 6 MEDLINE ON STN ACCESSION NUMBER: 93103619 MEDLINE DOCUMENT NUMBER: PubMed ID: 1466841

TITLE: Modulation of cell surface molecules during HIV-1 infection

of H9 cells. An immunoelectron microscopic study.

AUTHOR: Meerloo T; Parmentier H K; Osterhaus A D; Goudsmit J;

Schuurman H J

CORPORATE SOURCE: Department of Pathology, University Hospital, Utrecht, The

Netherlands.

SOURCE: AIDS (London, England), (1992 Oct) Vol. 6, No.

10, pp. 1105-16.

Journal code: 8710219. ISSN: 0269-9370.

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AB OBJECTIVE: To study cell surface molecules and HIV-1 proteins on H9 cells 2 days after infection by immunogold electron microscopy, either in single or in double labelling using combinations of host cell-derived molecules and HIV-1 proteins. DESIGN AND METHODS: The presence of host cell antigens CD3, CD4 and human leukocyte antigen-DR (HLA-DR) and HIV-1 antigens gag p15, p17, p24 and env gp41 was evaluated using immunocytochemistry at the light microscopic level. H9 cells 2 days after infection were processed for conventional transmission electron microscopy and cryo-ultramicrotomy. Leukocyte antigens investigated were CD2, CD3, CD4 (two antibodies), CD5, CD8, CD25, CD30, CD63 antigens and HLA-DR; HIV-1-encoded antigens were gag p24, pol reverse transcriptase, and env gp41 and gp120. Double immunogold labelling was performed using reagents with different sized gold particles. For leukocyte markers, the labelling density of the cell membrane was assessed quantitatively on uninfected and infected H9 cells. RESULTS: Infected cells revealed the presence of gag p24, pol, and env gp41 and gp120 antigens on HIV-1 virions. Uninfected H9 cells showed a random distribution of cell surface molecules, including CD4 antigen, along the plasma membrane. The CD63 antigen, a lysosomal membrane glycoprotein, was located mainly in the cytoplasm of uninfected cells. Cells 2 days after infection showed CD4 labelling on sites where virions were budding from or attached to the cell surface and on free virions. Virions also showed labelling by CD3, CD5, CD25, CD30 and CD63 antibodies and anti-HLA-DR. Compared with uninfected cells, a significantly lower density was found on infected cells in labelling for CD4, CD5 and anti-HLA-DR. A significantly higher density on cells 2 days after infection was seen in CD63 labelling. CONCLUSION: During the first phase of infection host cell molecules concentrate on budding structures and newly generated HIV-1 virions. This phenomenon might contribute to the disappearance of these molecules (like the CD4 molecule) from the cell membrane after infection.

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TITLE: Regulation of class II production after HIV-1 infection. AUTHOR(S): Kraus, T.; Chen, H.; Becker, K.; Rakoff, K. S.; Sperber, K.

CORPORATE SOURCE: Mt. Sinai Sch. Med., New York, NY 10029, USA SOURCE: FASEB Journal, (March 12, 1999) Vol. 13, No. 4

PART 1, pp. A292. print.

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